A Dissertation on

A STUDY ON METABOLIC SYNDROME IN YOUNG ISCHEMIC STROKE

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI

In partial fulfillment of the regulations
for the award of the degree of

M.D. (GENERAL MEDICINE)
BRANCH - I

KILPAUK MEDICAL COLLEGE
CHENNAI.

MARCH 2009

BONAFIDE CERTIFICATE

This is to certify that **"A STUDY ON METABOLIC SYNDROME IN YOUNG ISCHEMIC STROKE"** is a bonafide work done by **Dr.P.DHANALAKSHMI**, post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in partial fulfillment of regulations of **The Tamilnadu Dr.M.G.R.Medical University** for the award of **M.D.Degree Branch I (General Medicine)** during the academic period from May 2006 to March 2009.

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ETHICAL COMMITTEE OF
GOVERNMENT KILPAUK MEDICAL COLLEGE HOSPITAL
KILPAUK, CHENNAI-10.
Venue: Dean Chamber, Date: 3.1.2008

Chair person
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Director of Medical Education (OSD)
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TO WHOMSOEVER IT MAY CONCERN

Dear Sir / Madam

Sub: Internal Medicine – MD PG's Dissertation Ethical Committee – Reg.

Ref: Requisition from H.O.D. Medicine.

This is in reference to the letter dated 2.1.2008 regarding Ethical committee meeting clearance with regard to the following topics

Sl.No	Name of the Post Graduate	Dissertation Topic
1	Dr. R.Ramprasad	A study on Prevalence and Risk Factors of Diabetic Nephropathy in Newly Detected Type 2 Diabetic Patients
2	Dr. V.Sakthivadivel	A study on Cardiac abnormalities in HIV infected individuals
3	Dr. K.S.Gopakumar	A study on Subclinical Hypothyroidism in females over 50 years of age
4	Dr. T.Karthikeyan	Inflammatory profile in Acute Myocardial Infarction

5	Dr. Maliyappa Vijay Kumar	A study on Prevalence of Microalbuminuria in HIV patients not on ART and Correlation with CD4 count
6	Dr. D.Radha	High sensitivity C - reactive protein as a determinant in the outcome of Acute ischemic stroke
7	Dr. Lakshmi Thampy M.S	A Study on the prevalence of increased LV mass & Proteinuria in newly diagnosed Hypertensive patients.
8	Dr. Manu Bhasker	A study on the effect of Intravenous Metoprolol along with Thrombolysis in Acute Myocardial infarction
9	Dr. P.Mohanraj	Evaluation of Lipid Profile in patients with non diabetic Chronic Kidney Disease Stage 3,4 and 5
10	Dr. P.Dhanalakshmi	A study On Metabolic Syndrome in Young Ischemic Stroke
11	Dr. V.Murugesan	A study on Electrocardiographic and Echocardiographic changes in Chronic Obstructive Airway Disease
12	Dr. M.Seetha	Effect of Right ventricular infarction on the immediate prognosis of Inferior wall Myocardial Infarction

We are glad to inform you that at the EC meeting held on 3.1.08 on the above topics were discussed and **Ethically approved**.

DEAN

Chair person
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Chairman & Members of the Ethical Committee:

Chairman

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   Social Worker

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   Nursing Superintendent

We confirm that no member of the study team is on the Ethics Committee and no member of the study team voted.

The trial will also follow the Ethics Guidelines for Bio-Medical Research On Human subjects issued by ICMR, New Delhi and will not involve any expense to the Government and will not be detrimental to the normal functioning of the Institution.

The study will also satisfy the revised order issued by the Government of Tamil Nadu, Health and Family Welfare Department G.O.MS.No:319, H & FW, Dept. dated 30.11.2001.

INTRODUCTION

Stroke is the leading cause of adult disability and is the second commonest cause of death worldwide[1].More than two-thirds of the global burden of stroke is borne by developing countries, where the average age of patients with stroke is 15 years younger than in developed countries.[2] The available data indicate that stroke occurring in young people is more often atherothrombotic in origin in developing countries,[3-5] in contrast to the developed countries where arterial dissection and cardioembolic etiologies predominate.[6-9]

Ischemic stroke occurring in young Indians may be a manifestation of accelerated cerebrovascular atherosclerosis, paralleling the early age of onset of cerebrovascular diseases noted for this population.[10] Additionally based on prior data on increased propensity for insulin resistance among South Asians,[11,12] the ischemic stroke in young adults in India may be associated with the metabolic syndrome.

The term metabolic syndrome refers to a cluster of metabolic abnormalities related to a state of insulin resistance.[13]The characteristics

include insulin resistance, abdominal obesity, elevated blood pressure(BP), triglycerides(TG) and low levels of High density lipoprotein(HDL).

To assess the association between the metabolic syndrome (according to NCEP ATP III criteria)[14] and the acute ischemic non-embolic stroke a case control study was conducted, comparing 60 cases aged less than 45 years with a first ischemic (non embolic) stroke with 30 controls of same age group without prior history of stroke and heart disease.

Metabolic syndrome was diagnosed by using NCEP ATP III criteria except waist circumference. Cutoff point for abnormal waist circumference according to NCEP ATP III criteria is $\geq$102cm for males and $\geq$82cm for females. This may be fit for western population but it is very high for Indians. So the cutoff point for abnormal waist circumference was taken as per the new International Diabetes Federation(IDF) criteria where ethnic specific values were given. Cutoff point for South Asians as per the IDF criteria is >90cm for males and >80cm for females.

AIM OF THE STUDY

To assess the association between the metabolic syndrome and its individual components- fasting blood sugar (FBS), blood pressure (BP), HDL cholesterol(HDL-C), triglyceride(TG) and waist circumference(WC) and the first ischemic (non embolic) stroke in young adults below 45 years.

REVIEW OF LITERATURE

Stroke

Stroke or cerebrovascular accident is defined as an acute onset of focal neurological deficit that is attributed to a vascular cause which lasts for more than 24 hours. It may be due to atherosclerotic thrombosis or embolism or hemorrhage-aneurysm, AV malformation etc. If the deficit recovers fully within 24 hours it is named as transient ischemic stroke (TIA). Typically the signs and symptoms of a TIA last for 5 to 15 minutes.

Stroke is the leading cause of adult disability and the second commonest cause of death worldwide. More than two-thirds of the global burden of stroke is borne by developing countries.

20-30% strokes in India occurs < 45years. There is widely recognized race and ethnic disparity in stroke patients in general. Young Blacks and Hispanics are having increased morbidity and mortality from stroke compared to Whites.

Atherosclerosis remains the major cause not only for stroke but also for all vascular diseases. Moreover, current predictions estimate that by the

year 2020, atherosclerotic vascular diseases will become the leading global cause of total disease burden, defined as the years subtracted from healthy life by disability or premature death.

METABOLIC SYNDROME

Definition

Metabolic syndrome refers to a cluster of metabolic abnormalities related to a state of insulin resistance which is often associated with obesity. The major characteristics include insulin resistance, abdominal obesity, elevated blood pressure and lipid abnormalities (elevated levels of triglycerides and low levels of high density lipoprotein (HDL-C) cholesterol.

Metabolic syndrome is associated with proinflammatory and prothrombotic state that may include elevated levels of C- reactive protein, endothelial dysfunction, hyperfibrinogenemia, increased platelet aggregation increased levels of plasminogen activator inhibitor 1,elevated levels of uric acid, microalbuminuria and shift towards small, dense particles of low density lipoprotein cholesterol.

History

The term "metabolic syndrome" dates back to at least the late 1950s, but came into common usage in the late 1970s to describe various associations of risk factors with diabetes, that had been noted as early as the 1920s.[15,16]

- The Marseilles physician Dr. Jean Vague, in 1947, observed that upper body obesity appeared to predispose to diabetes, atherosclerosis, gout, and calculi.[17]
- Avogaro, Crepaldi and co-workers described six moderately obese patients with diabetes, hypercholesterolemia, and marked hypertriglyceridemia all of which improved when the patients were put on a hypocaloric, low carbohydrate diet.[18]
- In 1977, Haller used the term "metabolic syndrome" for associations of obesity, diabetes mellitus, hyperlipoproteinemia, hyperuricemia and steatosis hepatis when describing the additive effects of risk factors on atherosclerosis.[19]
- The same year, Singer used the term for associations of obesity, gout, diabetes mellitus, and hypertension with hyperlipoprotenemia.[20]

- In 1977 and 1978, Gerald B. Phillips developed the concept that risk factors for myocardial infarction concur to form a "constellation of abnormalities" i.e., glucose intolerance, hyperinsulinemia, hypercholesterolemia and hypertriglyceridemia and hypertension that is associated not only with heart disease, but also with aging, obesity and other clinical states. He suggested there must be an underlying linking factor, the identification of which could lead to the prevention of cardiovascular disease; he hypothesized that this factor was sex hormones.[21,22]
- In 1988, in his Banting lecture, Gerald M. Reaven proposed insulin resistance as the underlying factor and named the constellation of abnormalities as Syndrome X. Reaven did not include abdominal obesity, which has also been hypothesized as the underlying factor, as part of the condition.[23]

The terms "metabolic syndrome," "insulin resistance syndrome," and "Syndrome X" are now used specifically to define a constellation of abnormalities that is associated with increased risk for the development of type 2 diabetes and atherosclerotic vascular disease.

Prevalence of Metabolic Syndrome in India and the World

With the formulation of NCEP/ATP III guidelines, some uniformity and standardization has occurred in the definition of metabolic syndrome and has been very useful for epidemiological purposes. At present, metabolic syndrome is an all or none diagnosis.

The study results based on third National Health and Nutrition Examination Survey (NHANES III), showed that approximately one fourth of the US adults 20 years or older met the diagnostic criteria for metabolic syndrome.[24] The prevalence of the metabolic syndrome depends on age, ethnic background, and gender. It rises linearly from 20 to 50 years and plateaus thereafter.

Looking at various studies around the world, which included population samples, aged from 20 and onwards, the prevalence varies from 8% (India) to 24% (United States) in men and from 7% (France) to 46% (India) in women.[25]

Two Indian studies, which differed in their definition of obesity: first[26] used the obesity criteria suitable for Indians, while the second[27] used the standard ATP III definition of obesity. Both studies used population based samples within the age range but reported prevalence of 13% in Jaipur[27] and

41% in Chennai.[26] The prevalence of obesity in two study groups was quite similar (31% versus 33%), despite the different definitions used. Far larger differences were observed between the two studies for the prevalence of elevated triglycerides (46% vs. 30%), hypertension (55% vs. 39%) and elevated fasting plasma glucose (27% vs. 5%), each of which reported having used the same cut off points.

Interestingly, a third Indian study,[28] also from Chennai, reported a metabolic syndrome prevalence of 11.2% (Using EGIR criteria), which was much closer to the prevalence rate reported for Jaipur than the other Chennai study. Therefore, even within the same ethnic population group it appears that there can be significant differences in the prevalence of both the individual factors that constitute the metabolic syndrome and the metabolic syndrome itself.

Metabolic syndrome prevalence rates as described earlier vary among ethnic groups as defined by the ATP III criteria among Finnish and Native American men. Both studies involved subjects with comparable age ranges (42-60 and 44-49 years, respectively), with the Finnish study showing prevalence of only 14% compared with the prevalence in the Native American study of 43.6%. The prevalence varies from a low of 13.9% in black men to a high of 27.2% in Mexican American women.[29]

Causes of the metabolic syndrome

Insulin resistance

Insulin resistance and the metabolic syndrome appear to be almost inseparable. Insulin resistance was the basis of Reaven syndrome X and definitions of the metabolic syndrome required presence of insulin resistance. An elevated fasting blood glucose (used by some as a surrogate for insulin resistance) is one of the 5 components of the ATP III/AHA, NHLBI definition but any 3 conditions may be present for the diagnosis.

Obesity

Fat deposition in the upper body, and particularly intra-abdominal fat (visceral adiposity), has been thought to have greater metabolic consequences than fat on the hips or thighs, which is more common in women. Visceral adiposity is approximately 20% and 6% of total body fat in men and women, respectively. Unlike subcutaneous fat, blood circulation from visceral fat flows directly into the liver, thereby exposing it to high concentrations of free fatty acids and other chemicals and causing increased production of triglycerides and increased insulin resistance.

Obesity increases the risk of elevated fasting blood glucose or type 2 diabetes, high triglycerides, elevated blood pressure or hypertension, and low HDL cholesterol. At the present, almost 25% of adults in the United States (about 47 million people) have the metabolic syndrome. In non-Western countries such as India in which obesity is on the rise, the metabolic syndrome soon follows.

The individuals who have fallen as victims to the obesity epidemic do not necessarily have abdominal obesity. Therefore, it appears that abdominal obesity, while probably a great risk for the syndrome, is not an absolute requirement. Patients with normal blood glucose and weight who have an elevated blood pressure and triglycerides and a low HDL have, by definition, having the metabolic syndrome. While such individuals compose a small number of all those with the metabolic syndrome, it seems that insulin resistance or an elevated blood glucose are not necessary for the diagnosis.

Sedentary lifestyle

Sedentary lifestyle, that is, the lack of aerobic exercise is a major factor in the metabolic syndrome. But lack of exercise also tends to increase

weight, FBS, insulin resistance, blood pressure, and triglycerides. With sufficient daily physical activity, the metabolic syndrome would disappear.

Diet

Diet high in refined carbohydrates (sweets, regular soda, juice, white flour, white potatoes) indirectly promotes the metabolic syndrome and leads to weight gain, increased triglycerides, and a low HDL. Such diets are often high in sodium (salt), which increases the risk of high blood pressure.

Age

Aging increases the incidence of high blood pressure and elevated blood glucose; and older individuals tend to be less physically active (which leads to higher blood pressure and blood glucose).

Genetics

Metabolic syndrome in the South Indian population has recently been reported to be associated with Thr54 allele carriers of the Ala54Thr variant of FABP2 gene.

Others

Family history (Parents /siblings of DM 2)

Polycystic ovary syndrome

Pathogenesis

The mechanisms underlying the metabolic syndrome are not fully known; however insulin resistance plays a fundamental role in the pathogenesis of metabolic syndrome. Resistance to insulin stimulated glucose uptake seems to modify the biochemical responses in a way that predisposes to metabolic risk factors.[30,31] Insulin resistance causes lipolysis results in increased plasma free fatty acid. This promotes hepatic VLDL release and lowers HDL cholesterol. Insulin resistance is associated with impairment of endothelial nitric oxide system thus results in vasoconstriction. Increased activity of sympathetic nervous system noted in the metabolic syndrome causes hypertension.

A central role has been attributed to the pro-inflammatory cytokines, tumor necrosis factor alpha (TNF-alpha) and interleukin (IL)-6, that both are produced in substantial amounts by human adipose tissue. TNF- alpha impairs insulin-stimulated glucose uptake in a variety of cells and decreases lipoprotein lipase activity. Both cytokines increase hepatic lipogensis and elicit a systemic acute-phase response.[32] Increased hepatic release of

triglyceride rich VLDL results in insulin resistance by directly antagonizing insulin and inducing cytokines.

Various aspects of the acute-phase response, such as fibrinogen and plasminogen activator inhibitor-1 levels, whole-blood viscosity, and white blood cell count, have recently been found to correlate positively with the metabolic syndrome.[33] This is of particular interest because inflammation plays an important role in the pathogenesis of atherothrombosis.[35]

Macrophage and T-cell infiltration is a major feature of atherosclerotic plaques, especially at the sites of plaque rupture, and epidemiological studies show strong positive associations of systemic markers of inflammation with atherothrombotic disease[34,36,37,38] Moreover, C-reactive protein (CRP), the classic and exquisitively sensitive acute phase reactant, shows a strong independent association with the risk of Coronary Heart Disease and other atherothrombotic events. CRP levels have also been found to correlate with BMI and some features of the metabolic syndrome.

Insulin resistance and resulting hyperinsulinemia have been implicated in the development of type 2 diabetes, hypertriglyceridemia,

hypertension, polycystic ovary syndrome, hypercoagulability and vascular inflammation, as well as the eventual development of atherosclerotic cardiovascular disease manifested as myocardial infarction, stroke and end organ damage.

Signs and symptoms

- Fasting hyperglycemia — diabetes mellitus type 2 or impaired fasting glucose, impaired glucose tolerance, or insulin resistance
- High blood pressure
- Central obesity (also known as visceral, male-pattern or apple-shaped adiposity), overweight with fat deposits mainly around the waist
- Decreased HDL cholesterol
- Elevated triglycerides

Associated diseases and signs are: elevated uric acid levels, fatty liver (especially in concurrent obesity), progressing to non-alcoholic fatty liver disease, polycystic ovarian syndrome, hemochromatosis and acanthosis nigricans.

Diagnostic criteria

There are currently two major definitions for metabolic syndrome provided by the International Diabetes Federation (IDF) and the Revised National Cholesterol Education Program (NCEP) respectively. The revised NCEP and IDF definitions of metabolic syndrome are very similar and it can be expected that they will identify many of the same individuals as having metabolic syndrome. The two differences are that IDF excludes any subject without increased waist circumference, while in the NCEP definition metabolic syndrome can be diagnosed based on other criteria and the IDF uses geography-specific cut points for waist circumference, while NCEP uses only one set of cut points for waist circumference regardless of geography. These two definitions are much closer to each other than the original NCEP and WHO definitions.

WHO

The World Health Organization criteria (1999) require presence of diabetes mellitus, impaired glucose tolerance, impaired fasting glucose or insulin resistance and two of the following:

- blood pressure: ≥ 140/90 mmHg

- dyslipidaemia:

  triglycerides ≥1.695mmol/L(150mg/dl)

  high density lipoprotein cholesterol

  ≤ 0.9mmol/L (male)

  ≤ 1.0 mmol/L (female)

- central obesity

  waist: hip ratio > 0.90 (male); > 0.85(female) and/or

  body mass index > 30 kg/m^2

- microalbuminuria:

  urinary albumin excretion ratio ≥ 20 mg/min or

  albumin : creatinine ratio ≥ 30 mg/g

The WHO criteria differed from all subsequent definitions in two respects:

1) One of the risk factors was a measure of renal impairment based on the amount of protein excreted in the urine.

2) Obesity was determined either by waist-to-hip ratio or body mass index.

EGIR

The European Group for the Study of Insulin Resistance (EGIR) (1999) requires insulin resistance defined as the top 25% of the fasting insulin values among non-diabetic individuals and two or more of the following:

1) Central obesity:

waist circumference ≥ 94 cm (male), ≥ 80 cm (female)

2) Dyslipidaemia:

TG ≥ 2.0 mmol/L and /or

HDL-C < 1.0 mmol/L or

Treated for dyslipidaemia

3) Hypertension:

Blood pressure ≥ 140/90 mmHg or on antihypertensive medication

4) Fasting plasma glucose ≥ 6.1 mmol/L(110mg/dl)

NCEP(2001):

In 2001, led by Dr. Scott Grundy, the National Cholesterol Education Program Adult Treatment Panel (ATP III) was published and became what is now probably the most widely used definition of the metabolic syndrome in the United States. Neither the insulin resistance syndrome nor any other

single condition was a necessary component. The risk factor used as a surrogate for insulin resistance could be either DM-2 or an impaired fasting plasma glucose level (IFG).

The 2003 position statement by the American College of Endocrinology reasserted the primacy of insulin resistance and dropped the term metabolic syndrome. As in the earliest guidelines, insulin resistance was made a necessary component of the syndrome. A specific number of other risk factors were not included- DM-2 was not used in making the diagnosis and several clinical scenarios not included in other guidelines were added: family history of DM-2, ethic groups prone to DM, sedentary lifestyle, advancing age, and polycystic ovary syndrome (a condition often accompanied by insulin resistance and its associated abnormalities).

The US National Cholesterol Education Program Adult Treatment Panel III (2001) requires at least three of the following

- central obesity: waist circumference ≥ 102 cm or 40 inches (male), ≥ 88 cm or 36 inches(female)
- dyslipidaemia: TG ≥ 1.695 mmol/L (150 mg/dl)
- dyslipidaemia: HDL-C < 40 mg/dL (male), < 50 mg/dL (female)
- blood pressure ≥ 130/85 mmHg

- fasting plasma glucose ≥ 6.1 mmol/L (110 mg/dl)

The new International Diabetes Federation (IDF) definition

According to the new IDF definition, for a person to be defined as having the metabolic syndrome, he must have:

Central obesity

defined as waist circumference ≥ 94cm for Europid men and ≥ 80cm for Europid women, with ethnicity specific values for other groups plus any two of the following four factors:

- raised triglyceride level: ≥ 150 mg/dL (1.7 mmol/L), or specific treatment for this lipid abnormality
- reduced HDL cholesterol: < 40 mg/dL (1.0 mmol/L) in males and < 50 mg/dL (1.3 mmol/L) in females, or specific treatment for this lipid abnormality
- raised blood pressure: systolic BP ≥ 130 or diastolic BP ≥ 85 mm Hg, or treatment of previously diagnosed hypertension
- raised fasting plasma glucose (FPG) ≥ 100 mg/dL (5.6 mmol/L), or previously diagnosed type 2 diabetes

The IDF also delineated multiple, ethnic specific values for waist circumference and defined more stringent criteria for whites of European descent.

Europids:

The cut-off point for abnormal waist circumference decreased from 102 cm (40 inches) to 94 (37 inches) in men and from 88 cm (35 inches) to 80 (31.5 inches) for women. The IDF conceded that in the USA, the ATP III values (male 102 cm, female 88 cm) are likely to continue to be used.

Chinese and South Asians (based on Chinese, Malay, and Asian-Indian populations): male > 90 cm, female > 80 cm.

Japanese: male $\geq$ 85 cm, female $\geq$ 90 cm

2005 ATP III/AHA, NHLBI Diagnostic Criteria for the Metabolic Syndrome:

There is confusion as to whether the American Heart Association and National Heart, Lung, and Blood Institute intended to create another set of guidelines or simply update the NCEP ATP III definition. According to Scott Grundy, University of Texas Southwestern Medical School, Dallas,

Texas, the intent was just to update the NCEP ATP III definition and not create a new definition.[39]

Any 3 of the following 5 conditions fulfils the definition of the metabolic syndrome (using abnormal FBG as currently defined)

- Elevated waist circumference:

  Men - Equal to or greater than 40 inches (102 cm)

  Women - Equal to or greater than 35 inches (88 cm)

- Elevated triglycerides:

  Equal to or greater than 150 mg/dL

- Reduced HDL cholesterol:

  Men - Less than 40 mg/dL

  Women - Less than 50 mg/dL

- Elevated blood pressure:

  Equal to or greater than 130/85 mm Hg or use of medication for hypertension

- Elevated fasting glucose:

  Equal to or greater than 100 mg/dL (5.6 mmol/L) or use of medication for hyperglycemia

PARAMETERS TO DIAGNOSE METABOLIC SYNDROME

The definitions and criteria discussed earlier provide the parameters for the diagnosis of the syndrome:

- Fasting blood glucose (FBG)
- Fasting triglycerides
- HDL cholesterol

These three components of the metabolic syndrome require a fasting blood test for both the glucose and triglyceride levels; that is, nothing should be eaten or drunk other than water for 10 to 12 hours preceding the test.

- Blood pressure, measured by a clinician
- Waist circumference, measured by a clinician

Waist circumference measurement:

NHLBI recommendation for waist measurement is quite precise: place a tape measure at the top of the right iliac crest (pelvis), circle the abdomen on a horizontal plane parallel to the floor, make the tape snug without compressing the skin, and measure at the end of a normal expiration.

Other methods: measurement at the umbilicus, where the waist is narrowest; half-way between the last rib and the iliac crest; narrowest point between the costal margin and iliac crest.

Why is waist circumference used instead of body mass index?

The definition of the metabolic syndrome uses waist circumference as a surrogate for central obesity. Body mass index (BMI), a ratio of weight to height (weight in kilograms divided by height in meters square), provides no indication of the location of fat deposits. BMI is used to categorize the weight.

Body Mass Index Categories:

- Underweight: below 18.5
- Desirable weight: 18.5-24.9
- Overweight: 25-29.5
- Obese: 30-39.5
- Severely obese: 40 or higher

According to BMI categories, the overweight and obese reflect greater muscle mass and not fat (e.g. a body builder who develops a very large

muscle mass). Those with a BMI in the obese range may not have visceral obesity, and those with visceral obesity may have a BMI less than 30.

The waist-to-hip ratio, which was the WHO criterion for visceral obesity, is another measure of the same condition:

Normal values

women : less than 0.8;

men : less than 1.0.

Hip measurement is less controversial than that of waist and is usually measured at the maximum circumference above the buttocks.

Waist measurement indicates both too much fat and its central location. Central or upper body obesity probably poses a greater risk for CVD and DM-2. Central obesity is most accurately determined by imaging techniques such as direct measurement of visceral fat by computer assisted tomography or magnetic resonance imaging, which are expensive and impractical in clinical research.

Treatment for the Metabolic Syndrome

No specific treatment is recommended for the metabolic syndrome other than changes in lifestyle. Lifestyle changes would be promoted by

clinicians caring for individuals not only with diabetes and high blood pressure but also for those with mild increases that are now called prediabetes (fasting blood glucose 100 -125 mg/dL) and prehypertension (systolic blood pressure 130-139 or diastolic blood pressure (80-89).

Therapeutic Lifestyle modification

As the most frequent causes of the metabolic syndrome are obesity, sedentary lifestyle and poor diet, all the features of the metabolic syndrome will improve with lifestyle modification.[40]

- Weight loss

  Weight loss or more specifically decrease the percent body fat. Weight loss improves insulin sensitivity, increases HDL cholesterol and decreases FBG, blood pressure and triglycerides.

- Exercise [41-47]

  Frequent aerobic exercise will improve the same parameters as losing body fat. The current suggested minimum regimen is at least 30 minutes of brisk aerobic exercise (e.g. brisk walking) 4 days/week plus a modest amount of strength training. The optimal amount of aerobic exercise is 60 minutes 7 days/week.

- Diet

  a) Fat[48-51]

  Reduce the saturated fats.

  Total fat intake should be 25-35% of total calories.

  Low fat diets decrease triglycerides and increase HDL

  b) Carbohydrates [52,53]

  Reduce the processed food and refined carbohydrates (e.g. sugar, soft drinks, juice,desserts, white flour)

  Add whole grains, fruit, vegetables

  c) Protein

  Fish, preferably small fish low in mercury (sardines, herring, small mackerel, salmon)

- Reduce salt in diet[54,55]
- Reduce alcohol

  Alcohol increases appetite and triglycerides

Because the metabolic syndrome increases the risk of a CVD event, other risk factors for that condition should be addressed:

- Avoid Smoking
- Treatment for elevated LDL cholesterol

- Treatment for DM
- Treatment for high blood pressure

Medications

Obesity

Rimonaband- It is a cannabinoid-1 receptor antagonist.It inhibits intake of fatty foods.

Orlistat- It inhibits pancreatic lipase and reduces fat digestion.

Insulin Resistance

Weight reduction and increased physical activity will reduce insulin resistance.Two classes of drugs are currently available that reduce insulin resistance. These are metformin [56] and insulin sensitizers such as thiazolidinediones (TZDs).

Hyperglycemia

When patients with metabolic syndrome develop type 2 diabetes, they are at high risk for atherosclerotic vascular diseases.All risk factors should be intensively reduced. In addition, glucose levels should be appropriately treated with lifestyle therapies and hypoglycemic agents as needed to keep hemoglobin A1c levels below guideline targets.

Atherogenic dyslipidemia

Although statins typically are recognized to be LDL-lowering drugs, they reduce all apolipoprotein B–containing lipoproteins. Clinical studies demonstrate that abnormal lipoprotein patterns are doubly improved by combined statin-fibrate therapy.

Elevated Blood Pressure

Hypertensive patients with metabolic syndrome deserve lifestyle therapies to reduce blood pressure. In addition, antihypertensive drugs should be used as recommended by hypertension guidelines.

Prothrombotic state

No drugs are available that target PAI-1 and fibrinogen. An alternative approach to the prothrombotic state is antiplatelet therapy. For example, low-dose aspirin reduces CVD events in both secondary and primary prevention. Thus, use of aspirin for primary prevention in patients with metabolic syndrome is promising.

ATP III Guidelines for the Treatment of Patients with Metabolic Syndrome

Targeted area	Goal
Treat LDL cholesterol first.	
CHD and CHD risk equivalent (10-year risk for CHD >20 percent)	<100 mg per dL (<2.6 mmol per L)
At least two risk factors (10-year risk <=20 percent)	<130 mg per dL (<3.35 mmol per L)
Institute weight control.	10 percent from baseline
Institute physical activity.	30 to 40 minutes per day
Monitor treatment of hypertension.	<130/85 mm Hg
Treat elevated triglyceride levels and low HDL cholesterol levels	
Goal of non-HDL cholesterol for patients with triglyceride levels of ≥ 200 mg per dL	High CHD risk: <130 mg per dL Intermediate CHD risk: <160 mg per dL Low CHD risk: <190 mg per dL
ATP = Adult Treatment Panel; LDL = low-density lipoprotein; CHD = coronary heart disease; HDL = high-density lipoprotein	

MATERIALS AND METHODS

Study site

This study was conducted in Government Royapettah Hospital, Chennai during the period of 2006- 2008.

Study Population:

Cases:

60 patients aged less than 45 years hospitalized with the history of first ischemic stroke were included.

Inclusion Criteria:

1. Subjects aged less than 45 years.
2. Presented with first acute ischemic stroke-CT or/and MRI brain was taken for confirmation of ischemic stroke.

Exclusion Criteria:

1. Prior history of Stroke
2. Hemorrhagic stroke
3. Presentation to the hospital more than 6 months after the stroke onset.

4. Embolic stroke – complete cardiac evaluation & carotid Doppler was done to rule out embolic stroke.
5. Stroke due to intra cranial sinus thrombosis

Controls:

30 subjects from a community based sample of individuals aged less than 45 years without prior history of stroke and heart disease were included.

Data collection:

Data collection was done by using a structured proforma. BP, fasting blood sugar, fasting lipids were taken at approximately a week or later after stroke onset, as elevations in BP, blood sugar and lipids are well documented during the acute phase of stroke.[57,58]Waist circumference was measured at the highest point of iliac crest at the end of normal expiration.

RESULTS AND ANALYSIS

Characteristics of study population were assessed and the values were expressed as mean ± standard deviation except for age, sex and smoking. Prevalence of the individual risk factors and the metabolic syndrome in both groups were compared.

The association between the metabolic syndrome and its individual components and stroke was determined by the logistic regression analysis. Odds ratio and their 95% confidence intervals were assessed.

Significance was defined as P value <0.05 in all cases.

Characteristics of the study population

Stroke patients had high level of fasting blood sugar, BP, total cholesterol, triglycerides and increased waist circumference compared to control group.

Stroke was common in males.73% stroke patients were males. Stroke was more prevalent in 40-45 yrs age group. 53% of stroke patients were in

the age group of 41-45yrs. 60% of stroke patients had metabolic syndrome and 10% of control group had metabolic syndrome.

Table no 1

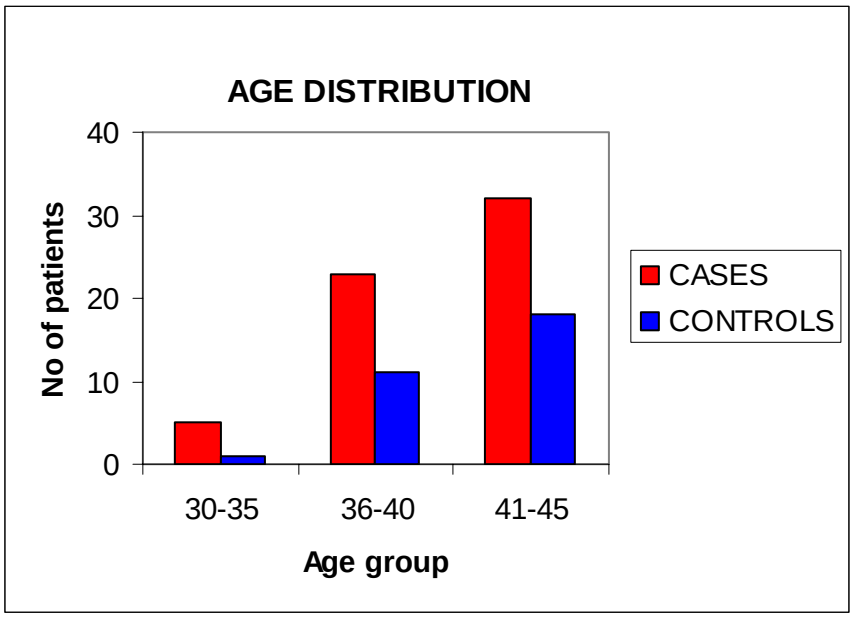
CHARACTERISTIC FEATURES OF STUDY POPULATION

Sl.no	Characterestic feature		Cases n – 60	Control n – 30	P value
1	Gender	Male	44[73.30%]	24[80.00%]	
		Female	16[26.70%]	06[20.00%]	
2	Age group	30-35	05[08.30%]	01[03.30%]	
		36-40	23[38.30%]	11[36.70%]	
		41-45	32[53.30%]	18[60.00%]	
3	Smoking		33[55.00%]	08[26.7%]	<0.01
4	FBS		115.73±28.11	90.73±18.30	<0.01
5	Syst BP		127.00±16.19	116.00±11.92	<0.01
6	Dias BP		81.58±08.90	78.17±08.90	0.07
7	HDL		42.62±08.44	46.63±09.12	0.04
8	Total cholesterol		196.80±59.35	164.90±40.78	0.01
9	Triglycerides		156.70±53.09	132.60±39.85	0.03
10	W.C		82.53±11.43	80.40±20.27	0.53

Table no 2

AGE DISTRIBUTION

AGE GROUP	CASES	CONTROLS	TOTAL
30-35	05	01	06
36-40	23	11	34
41-45	32	18	50
TOTAL	60	30	90

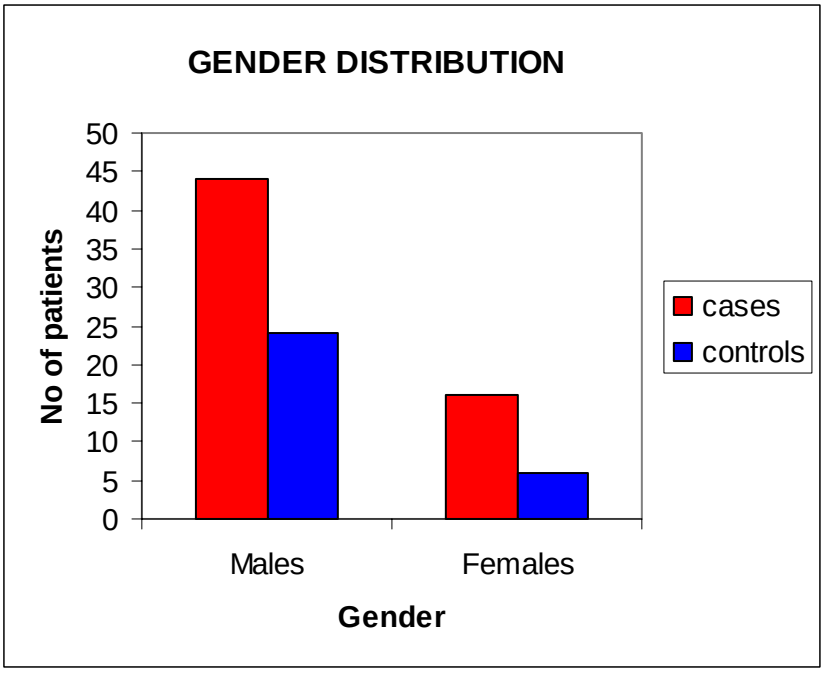


53.3% of stroke patients were in the age group of 41-45yrs

Table no 3

GENDER DISTRIBUTION

GENDER	CASES	CONTROLS	TOTAL
MALES	44	24	68
FEMALES	16	06	22
TOTAL	60	30	90

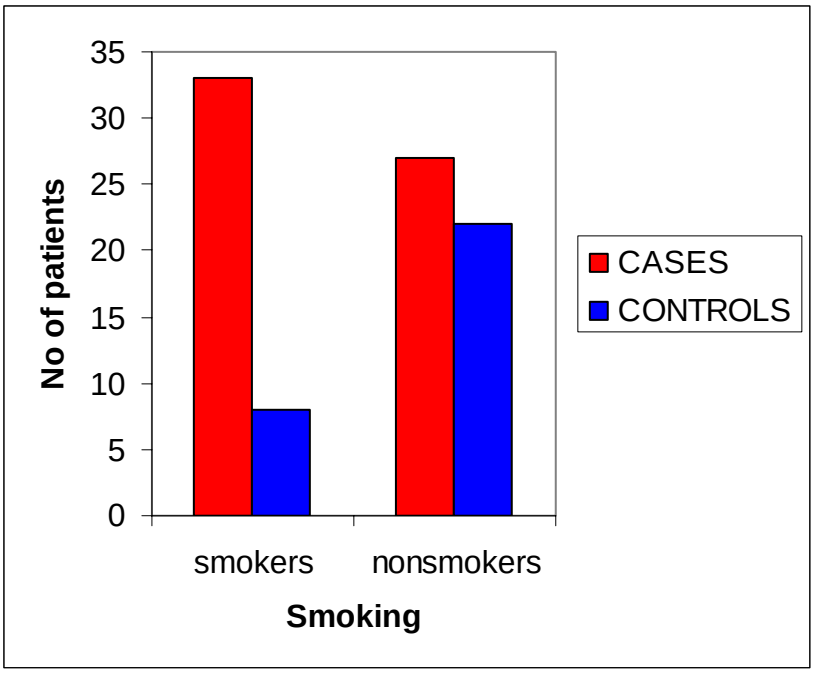


73.3% of stroke patients were males

Table no 4

PREVALENCE OF SMOKING

SMOKING	CASES	CONTROLS	TOTAL
SMOKERS	33	08	41
NON SMOKERS	27	22	49
Total	60	30	90



55% of stroke patients were smokers.

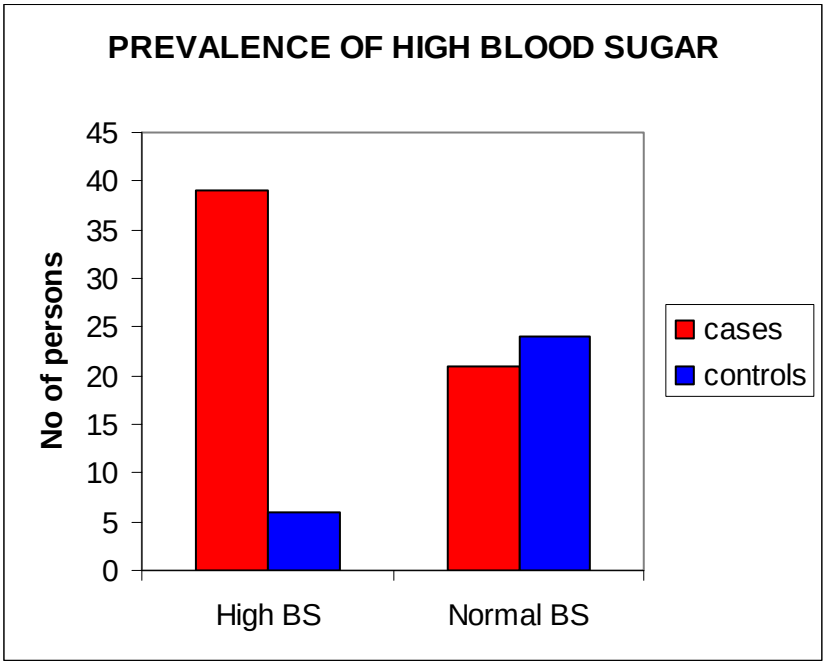
p value 0.01

Table no 5

PREVALENCE OF HIGH BLOOD SUGAR

[FBS ≥100mg/dl or diabetic on treatment]

BLOOD SUGAR	CASES	CONTROLS	TOTAL
HIGH BS	39	06	45
NORMAL BS	21	24	45
TOTAL	60	30	90



65% of stroke patients had high blood sugar.

20%of control group had high blood sugar.

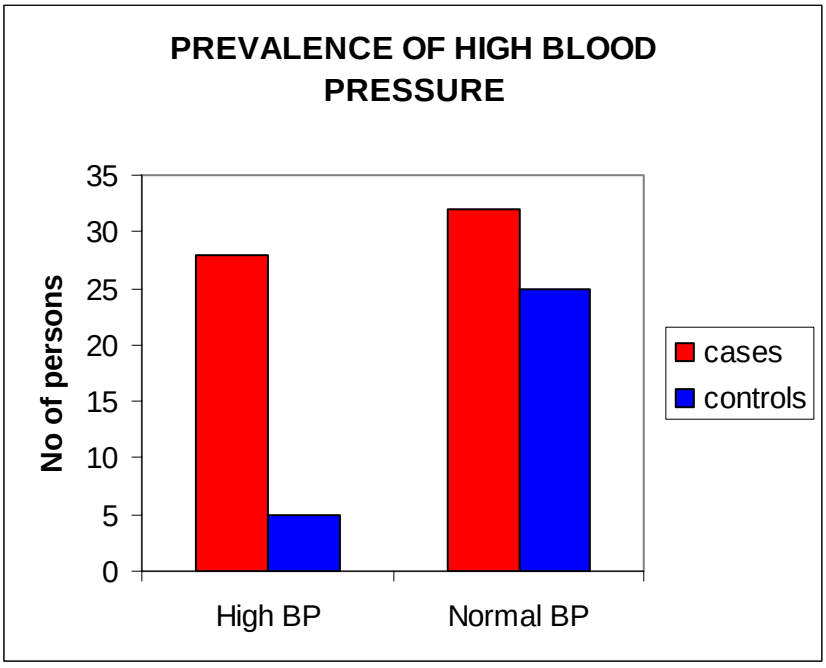
P value <0.001

Table no 6

PREVALENCE OF HIGH BLOOD PRESSURE

[BP ≥130/85mmHg or on anti hypertensive]

BLOOD PRESSURE	CASES	CONTROLS	TOTAL
High BP	28	05	33
Low BP	32	25	57
Total	60	30	90



46.7% of stroke patients had high BP.

16.7% of control group had high BP.

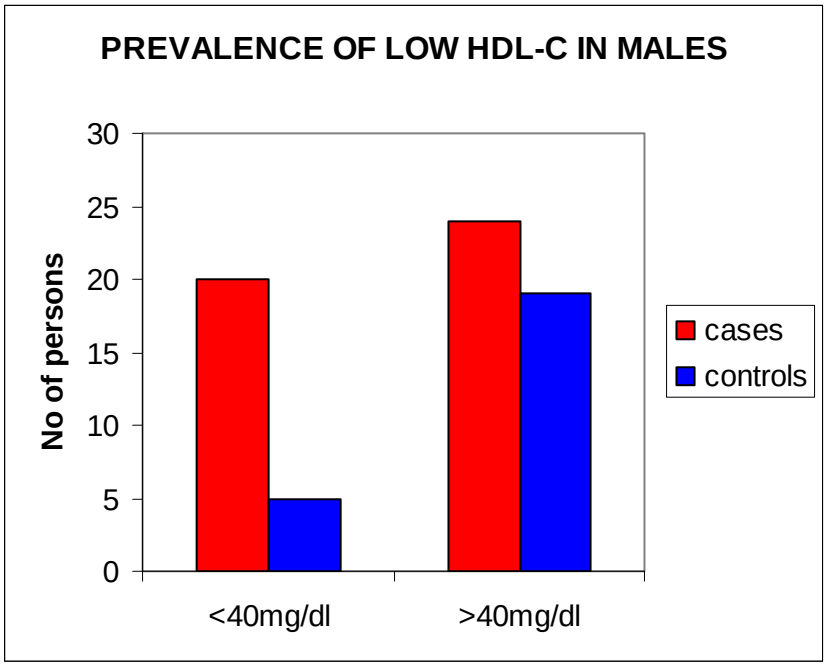
P value 0.005

Table no 7

PREVALENCE OF LOW HDL-C IN MALES

[Males <40mg/dl]

HDL	CASES	CONTROLS	TOTAL
<40mg/dl	20	05	25
>40mg/dl	24	19	43
Total	44	24	68



45.5% of stroke patients had low HDL.

20.8% of control group had low HDL.

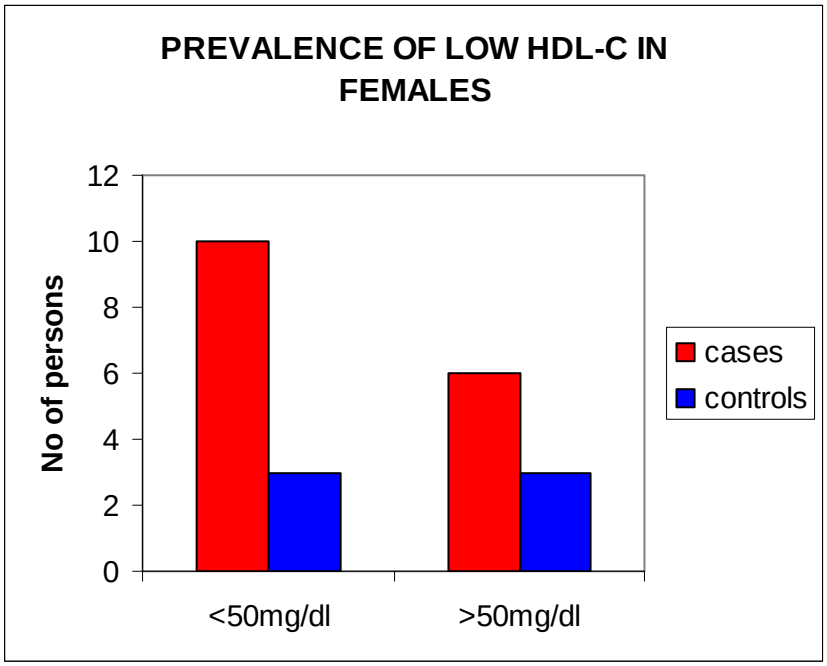
P value 0.04

Table no 8

PREVALENCE OF LOW HDL-C IN FEMALES

[Females <50mg/dl]

HDL	CASES	CONTROLS	TOTAL
<50mg/dl	10	03	13
>50mg/dl	06	03	09
Total	16	06	22



62.5% of stroke patients had low HDL.

50% of control group had low HDL.

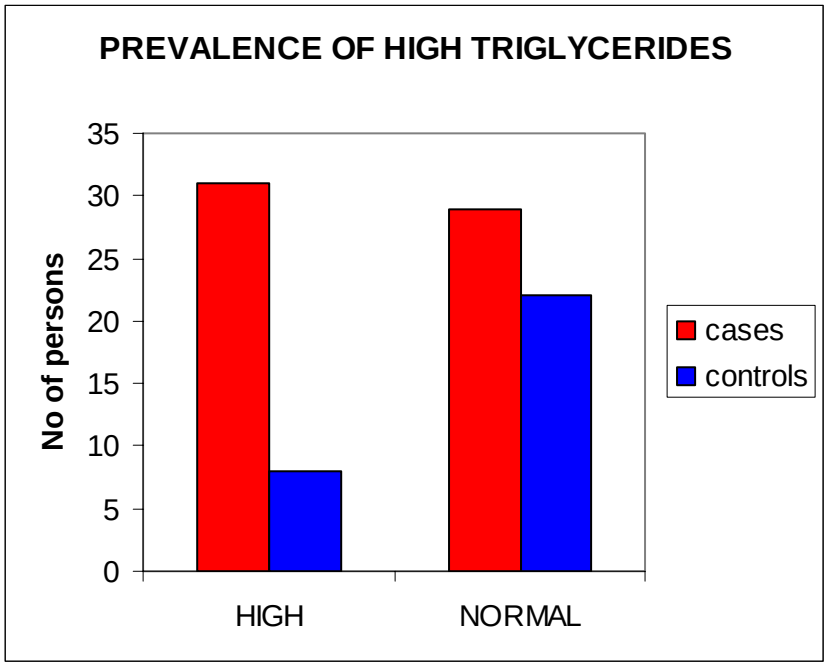
P value is not significant.

Table no 9

PREVALENCE OF HIGH TRIGLYCERIDES

[TG≥150mg/dl]

TG	CASES	CONTROLS	TOTAL
HIGH	31	08	39
NORMAL	29	22	51
Total	60	30	90



51.7% of stroke patients had high TG.

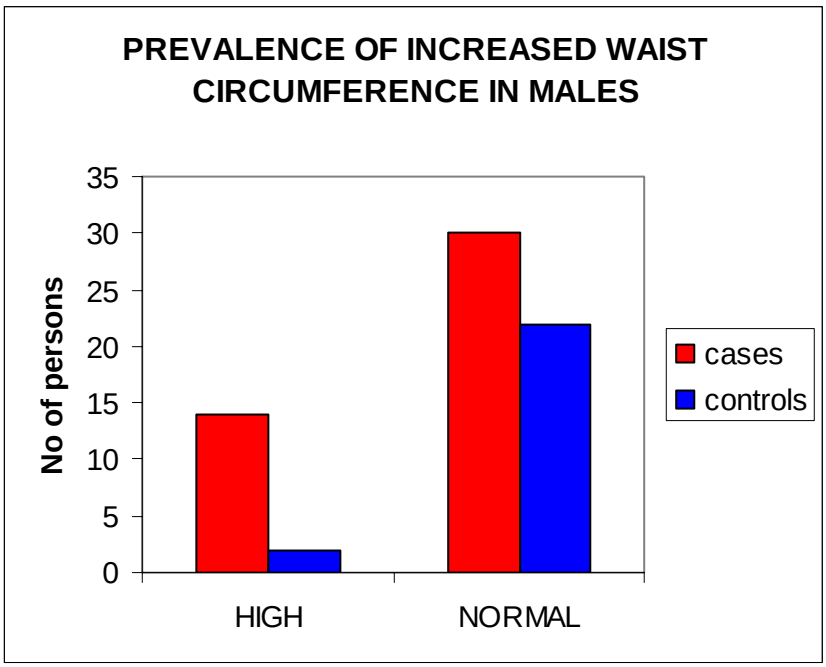
26.7% of control group had high TG.

P value 0.02.

Table no 10

PREVALENCE OF INCREASED WAIST CIRCUMFERENCE IN MALES [>90cm for South Asian males according to IDF criteria]

WAIST CIRCUMFERENCE	CASES	CONTROLS	TOTAL
HIGH	14	02	16
NORMAL	30	22	52
Total	44	24	68



31.8% of stroke patients had increased waist circumference.

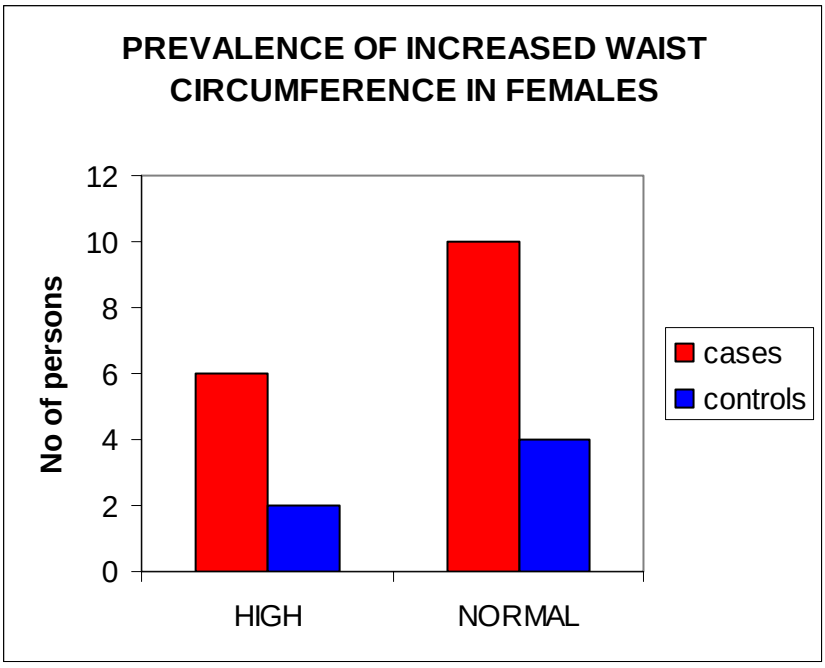
8.3% of control group had increased waist circumference.

P value 0.03.

Table no 11

PREVALENCE OF INCREASED WAIST CIRCUMFERENCE IN FEMALES [>80cm for South Asian females according to IDF criteria]

WAIST CIRCUMFERENCE	CASES	CONTROLS	TOTAL
HIGH	06	02	08
NORMAL	10	04	14
TOTAL	16	06	22



37.5% of stroke patients had increased waist circumference.

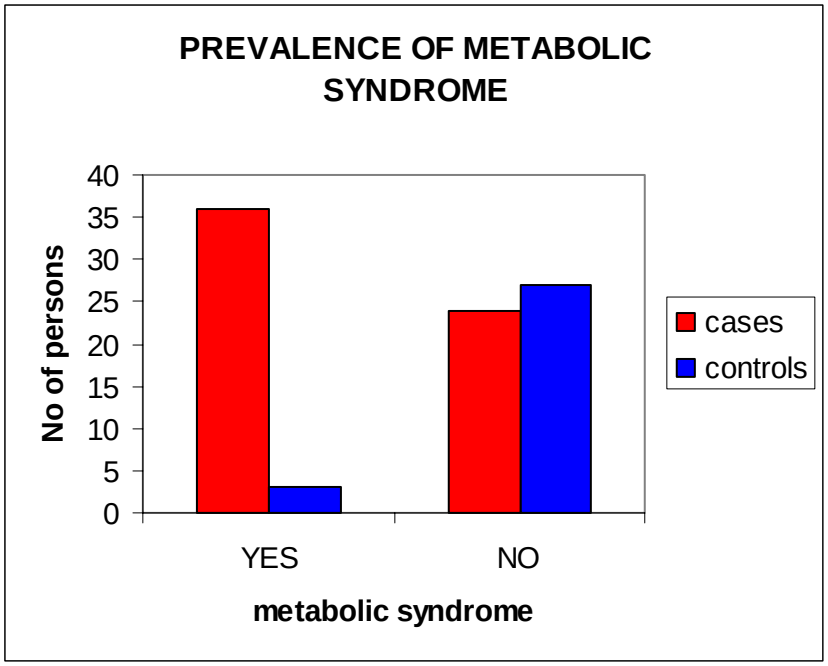
33.3% of control group had increased waist circumference.

P value is not significant.

Table no 12

PREVALENCE OF METABOLIC SYNDROME

METABOLIC SYNDROME	CASES	CONTROLS	TOTAL
Present	36	03	39
Absent	24	27	51
Total	60	30	90



60% of stroke patients had metabolic syndrome.

10% of control group had metabolic syndrome.

P value <0.01

Table no 13

LOGISTIC REGRESSION ANALYSIS

Association between the metabolic syndrome and its components and the acute ischemic stroke

Sl.no	Features	Odds ratio	95% CI	P value
1	**FBS≥100mg/dl**	4.05	1.810-8.84	0.01
2	**Syst BP≥130mmHg**	1.08	0.968-1.205	0.17
3	**Diast BP≥85mmHg**	1.02	0.848-1.221	0.85
4	**Low HDL**	2.18	1.01-4.011	0.03
5	**TG≥150mg/dl**	1.00	0.990-1.022	0.38
6	**High WC**	1.04	0.996-1.090	0.73
7	**Metabolic syndrome ≥3**	9.60	2.022-19.97	0.01
8	**Smoking**	8.01	0.97-13.02	0.02

DISCUSSION

In this case control study, stroke patients had higher fasting blood sugar, BP, abnormal lipid profile and increased waist circumference compared to community controls. Stroke was more prevalent in the age group of 41-45yrs. 53.3% of stroke patients belong to 41-45 yrs. Incidence of the stroke increases as the age advances. Stroke was more prevalent in males than females. 73.3% of stroke patients were males and 26.7% were females.

FBS was higher in stroke group than control group (115.73 VS 90.73, P value <0.01). Higher FBS was associated with 4 fold stroke risk (4 odds, P value 0.01) compared to control group and statistically significant. Insulin resistance is the pathophysiological process underlying the clustering of vascular risk factors in the metabolic syndrome.

BP was higher in stroke group than control group. The presence of hypertension was associated with increased risk of acute ischemic stroke. However, this association was not statistically significant because of the high rate of hypertension in the control group. It is well-documented that the

prevalence of hypertension rises with advancing age. For example, in the Framingham cohort study,[59]aged 70 to 79 almost 50% had borderline hypertension. Among metabolic syndrome components, hypertension is considered the least "metabolic. It is multifactorial in origin, with increasing arterial stiffness significantly contributing to systolic hypertension in the elderly.

Dyslipidemia is a hallmark of the Metabolic Syndrome. Stroke patients had low HDL-C, high total cholesterol (TC) to HDL-C ratio and high triglyceride. HDL-C level was inversely related to the risk of ischemic stroke. The protective role of HDL-C was attenuated in the presence of metabolic syndrome. In a case control study involving 204 patients with acute ischaemic stroke of all ages from Madras, South India, the authors found that while low HDL cholesterol and high total cholesterol to HDL cholesterol ratio were more frequent among patients.[60]

Abdominal obesity in association with metabolic syndrome carries high risk for stroke. Visceral fat is associated with insulin resistance than any other adipose tissue compartment. In this study stroke patients had

increased WC than the control group. But association with stroke risk was not statistically significant.

According to NCEP-ATP III presence of three or more components is defined as metabolic syndrome. In this study 60% of stroke patients had metabolic syndrome and it was associated with nine fold stoke risk which was statistically highly significant. (9 Odds, P value 0.01)

Smokers were associated with eight fold increased risk for stroke than nonsmokers which is statistically highly significant.(8 Odds, P value 0.02).Smoking is an independent risk factor for stroke.

In this study, high fasting blood sugar, low HDL cholesterol, smoking and metabolic syndrome were associated with increased stroke risk which is statistically highly significant.

A case control study, conducted in 214 South Indian patients aged between 15-45 years with first acute ischemic stroke in Sri Chitra Trunal Institute, Trivandrum[61] showed high fasting blood sugar, hypertension and smoking were associated with increased stroke risk. High

FBS associated with 4 fold stroke risk; smoking associated with 8 fold stroke risk; Metabolic syndrome associated with 6 fold stroke risk than community control group.

The two case control studies from India that included ischemic stroke in all age groups suggested that Hypertension, Diabetes mellitus and smoking are important risk factors.[62]

In the Baltimore–Washington Cooperative Young Stroke Study,[63] which compared 296 cases of incident ischaemic stroke among black and white adults aged 18–44 years with 1220 community based adults of the same age group, hypertension, diabetes mellitus and current smoking emerged as important risk factors.

Melbourne Risk Factor Study,[64] where 201 patients with first onset stroke due to cerebral infarction aged 15–55 years compared with the same number of matched neighbourhood control subjects showed hypertension, diabetes mellitus, current smoking, heart disease and long term heavy alcohol consumption were major risk factors. Although observational studies

from Western countries have emphasised the preponderance of cardiogenic embolism, arterial dissection, procoagulant states and non-atherosclerotic vasculopathies as possible aetiologies, careful analytic comparisons have shown the importance of traditional risk factors in the pathogenesis of stroke in the young adults.

SUMMARY

1. Prevalence of stroke increases as the age advances.
2. Stroke was more prevalent in males than females.
3. Stroke patients had high fasting blood sugar, high BP, elevated total cholesterol, triglyceride, low HDL-C and increased waist circumference than control group.
4. Metabolic Syndrome was more prevalent in stroke patients. 60% of stroke patients had metabolic syndrome ; 10% in control group.
5. High FBS was associated with 4 fold stroke risk (4 odds, p 0.01) compared to control group.
6. Low HDL-C was associated with 2 fold stroke risk (2 odds, p 0.03) compared to control group.
7. Metabolic Syndrome was associated with 9.6 fold stroke risk (9.6 odds, p 0.01) compared to control group.
8. Smoking was associated with 8 fold stroke risk (8 odds, p 0.02) compared to control group.

CONCLUSION

1) Metabolic syndrome and its individual components (high FBS and low HDL-C) were associated with increased risk for ischemic stroke in young adults.

2) Smoking is an independent risk factor for ischemic stroke.

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ABBREVIATION

1. FBS-fasting blood sugar
2. BP-blood pressure
3. HDL-C-high density lipoprotein cholesterol
4. TC-total cholesterol
5. TG-triglycerides
6. WC-waist circumference
7. NCEP ATP III-National Cholesterol Education Programe Adult Treatment Panel III
8. IDF-International Diabetes Federation
9. NHLBI-National Heart, Lung,Blood Institute

PROFORMA

Name: Age/Sex: Occupation:

Address: I.P NO:

Contact No: D.O.A:

Ref Dr: D.O.D:

Chief complaints:

Presenting illness:

Past History:

Treatment History:

Personal History:

Family History:

General Examination:

Height:

Weight:

W.C.:

Anemia:

Jaundice:

Clubbing:

Cyanosis:

Lymphadenopathy:

Pedal Edema:

Vitals:

Temp:

Pulse:

BP:

RR:

JVP:

Systemic examination:

Respiratoy System:

Cardiovascular system:

Abdomen:

Central nervous system:

Investigations:

1. Complete Hemogram
2. Fasting blood sugar
3. Renal parameters – Blood urea, Serum Creatinine, Serum Electrolytes.
4. Fasting Lipid profile
5. ECG
6. CXR
7. CT Brain / MRI Brain
8. Echocardiogram
9. Carotid Doppler Study

MASTER CHART

Sl no	Group	Name	Gender	FBS	DM	BP	HT	HDL	TG	WC	MS	Smoking
1	1	KUMAR	1	0	0	0	0	1	0	0	0	1
2	1	RAJESH	1	0	0	1	0	0	1	0	0	1
3	1	VIMALA	2	1	1	0	0	1	0	0	0	0
4	1	NAVEEN	1	0	0	0	0	0	0	0	1	1
5	1	SUMAN	1	1	1	1	0	1	0	1	1	1
6	1	GEETHA	2	1	1	0	0	1	1	1	1	0
7	1	GOKUL	1	1	1	1	1	1	1	0	1	0
8	1	HARISH	1	0	0	0	0	0	1	0	0	1
9	1	ARUN	1	1	0	1	1	1	1	0	1	1
10	1	SUDHA	2	1	1	1	0	1	0	0	1	0
11	1	MOHAMMED	1	1	1	0	0	1	0	1	1	0
12	1	MOHAN	1	0	0	0	0	0	0	0	0	1
13	1	NANDHINI	2	0	0	1	1	1	1	0	1	0
14	1	DINESH	1	0	0	0	0	0	1	0	0	1
15	1	RAMESH	1	1	1	0	0	1	1	0	1	1
16	1	GUNASEKAR	1	0	0	1	0	1	0	0	0	1
17	1	GANESH	1	0	0	1	0	0	1	0	0	1
18	1	ANTONY	1	1	1	0	0	1	0	1	1	1
19	1	GANGA	2	0	0	1	0	1	1	0	1	0
20	1	MANI	1	1	1	0	0	0	0	0	0	1
21	1	YAMUNA	2	1	1	0	0	1	1	0	1	0

Sl no	Group	Name	Gender	FBS	DM	BP	HT	HDL	TG	WC	MS	Smoking
22	1	ARUL	1	1	0	1	1	1	1	0	1	0
23	1	PRASATH	1	0	0	0	0	0	0	0	0	1
24	1	KALYANI	2	1	0	1	1	1	1	0	1	0
25	1	MUNUSAMY	1	1	0	1	1	1	0	0	1	0
26	1	RAMASAMY	1	1	0	0	0	1	0	1	1	1
27	1	SUDAR	1	0	0	0	0	0	0	0	0	1
28	1	JAGAN	1	0	0	1	0	1	1	0	1	0
29	1	SUNDARI	2	1	0	0	0	0	1	0	0	0
30	1	SIVAJI	1	1	1	0	0	1	1	0	1	1
31	1	KANNAN	1	1	1	0	0	1	1	0	1	1
32	1	SANKAR	1	0	0	0	0	0	0	1	1	1
33	1	JAYANTHI	2	1	0	1	1	0	1	1	1	0
34	1	SHAMEEM	1	0	0	0	0	0	0	0	0	1
35	1	SASIKUMAR	1	1	1	0	0	1	1	1	1	1
36	1	JAMES	1	1	0	1	1	1	0	0	0	1
37	1	VINOTH	1	1	1	1	0	1	0	0	0	0
38	1	KUMUDHA	2	0	0	0	0	1	1	1	1	0
39	1	HUSSAIN	1	1	0	1	1	0	0	1	1	0
40	1	LALITHA	1	1	0	0	0	1	1	0	1	0
41	1	SELVAN	1	1	1	1	0	0	1	0	1	1
42	1	FATHIMA	2	0	0	0	0	1	1	1	1	0
43	1	MARY	2	1	1	0	0	0	0	0	0	0
44	1	KRISHNAN	1	0	0	1	0	0	0	1	0	1

Sl no	Group	Name	Gender	FBS	DM	BP	HT	HDL	TG	WC	MS	Smoking
45	1	SABAPATHY	1	1	0	1	0	0	0	0	0	1
46	1	SUMATHI	2	1	0	1	1	0	1	0	1	0
47	1	BOOPATHY	1	1	0	1	0	0	1	1	1	0
48	1	GANAPATHY	1	0	0	0	0	0	0	0	0	1
49	1	MURUGAN	1	1	0	0	0	0	0	1	0	0
50	1	VIJAY	1	1	0	0	0	0	0	1	0	1
51	1	LOGANADHAN	1	1	0	1	0	1	0	0	1	0
52	1	JOHN	1	1	0	1	1	1	0	0	1	1
53	1	BALA	1	0	0	0	0	0	1	1	0	1
54	1	KUMARAN	1	0	0	0	0	0	0	0	0	1
55	1	SRIKUMARAN	1	1	0	0	0	0	1	1	1	0
56	1	GOMADHI	2	1	0	1	0	0	0	0	0	0
57	1	AMALA	2	1	0	1	1	0	1	1	1	1
58	1	STEPHEN	1	1	0	1	0	0	1	0	1	1
59	1	NARMADHA	2	1	0	0	0	1	1	1	1	0
60	1	SEENU	1	1	0	0	0	0	1	1	1	1

Sl no	Group	Name	Gender	FBS	DM	BP	HT	HDL	TG	WC	MS	Smoking
1	0	DIVYA	2	0	0	0	0	0	0	1	0	0
2	0	FEDRIK	1	0	0	0	0	0	0	0	0	0
3	0	GOKUL	1	0	0	0	0	1	0	0	0	0
4	0	VISU	1	0	0	0	0	0	0	0	0	0
5	0	KAMALI	2	1	1	0	0	0	0	0	0	0
6	0	NARESH	1	0	0	1	1	0	0	0	0	0
7	0	ANAND	1	0	0	0	0	0	1	0	0	0
8	0	PARUTHI	1	1	0	0	0	1	1	0	1	0
9	0	JACK	1	1	0	0	0	0	1	0	0	0
10	0	BAGAT SINGH	1	0	0	0	0	0	0	0	0	0
11	0	ANURADHA	2	0	0	1	0	1	0	0	0	0
12	0	VELU	1	0	0	9	0	0	0	1	0	0
13	0	RAJA	1	0	0	9	0	0	0	0	0	1
14	0	RATHNAM	1	0	0	9	0	0	0	0	0	0
15	0	SHANMUGAN	1	0	0	9	0	0	0	0	0	1
16	0	MATHI	1	0	0	9	0	0	0	0	0	0
17	0	NALINI	2	1	1	1	1	1	1	0	1	1
18	0	AZHAGAN	1	0	0	0	0	1	0	0	0	0
19	0	ASOK	1	1	0	0	0	0	1	0	0	1

Sl no	Group	Name	Gender	FBS	DM	BP	HT	HDL	TG	WC	MS	Smoking
20	0	VIVEK	1	0	0	0	0	0	0	0	0	1
21	0	RUPA	2	0	0	1	0	1	1	0	1	0
22	0	NAGARAJ	1	0	0	0	0	0	0	0	0	1
23	0	RAMAN	1	0	0	0	0	0	0	0	0	0
24	0	PRIYA	2	0	0	0	0	0	0	1	0	0
25	0	THILAK	1	1	1	0	0	0	1	0	0	0
26	0	SEETHARAM	1	0	0	0	0	0	0	0	0	0
27	0	RAJARAMAN	1	0	0	0	0	0	0	0	0	1
28	0	BARANI	1	0	0	1	1	1	1	0	0	1
29	0	GNANASUNDAR	1	0	0	0	0	1	0	0	0	0
30	0	PALANI	1	0	0	0	0	0	0	1	0	0

KEY TO MASTER CHART

Group

1- cases

2- controls

Gender

1- males

2- females

Age group

1- 31-35yrs

2- 36-40yrs

3- 41-45yrs

FBS-fasting blood sugar

0- <100mg/dl

1- ≥100mg/dl

DM-Diabetes Mellitus

0- not a known diabetic

1- known diabetic on treatment

BP-Blood Pressure

0- <130/85mmHg

1- ≥130/85mmHg

HT-Hypertension

0- not a known hypertensive

1- known hypertensive on treatment

HDL- high density lipoprotein

0- >40mg/dl in males

>50mg/dl in females

1- ≤40mg/dl in males

< 50mg/dl in females

TG-triglycerides

0- <150mg/dl

1-≥150mg/dl

WC-waist circumference

0-<90cm in males

< 80cm in females

1- >90cm in males

>80cm in females

MS-metabolic syndrome

0- absent

1- present

Smoking

0- absent

1- present